ACTINOMYCOSIS OF THE TONSILS,

WITH SOME REMARKS ON THE PORTALS OF INFECTION.

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The only report I can find in literature of the lesion in this situation is by Ruge, in 1896,[1] a member of Gerhardt's clinic. He described four cases, which, wonderful to say, he discovered by the microscopic examination of only twenty-five tonsils. He suggested that systematic examinations of excised tonsils would more frequently reveal the condition. On a reference to my index I find that I have examined microscopically hypertrophied faucial tonsils from more than seventy-five cases. This is the only one in which an actinomycotic clump was present in the sections. It is possible of course that if the whole of every tonsil was reduced to sections, something of Ruge's prediction might have been observed. The clumps, or, as the Germans call them, the "drusen," are not to be mistaken by anyone accustomed to histological work. In a search through the *Centralblatt für Laryngologie* since its first number, a matter of twenty years, only one other reference was found to the existence of a local lesion of actinomycosis in the tonsil, though abscesses and infiltrations around the jaws and neck are cited in great abundance. With the report of this case I am familiar only in references,[2] it having been published originally by Lesin in the Russian *Wratsch*. This latter case died of cerebral involvement and a focal abscess was found in one tonsil and a scar in the other. Ruge concluded, from the fact that his cases presented no lesion elsewhere and had no subsequent trouble after excision of the tonsils, they could only be something that resembled actinomycosis, and reported them under the title "Ueber Actinomyces-ähnliche Gebilde," etc.

Therefore, if rarity of occurrence is any excuse for the publication of this case, I might be justified on that ground alone, but a "rare case" which fits into no category and teaches no lesson furnishes idle reading and is a futile labor for the author. The fewer such facts a man has to put in his mental storehouse the better.

It is my interest in the suggestiveness of this case which induces me to bring it before you for consideration. Incidentally it illustrates one point which I have attempted to emphasize on many occasions, and that is the desirability of examining microscopically absolutely all tissue removed from the nose and throat in the routine of either hospital or private practice.

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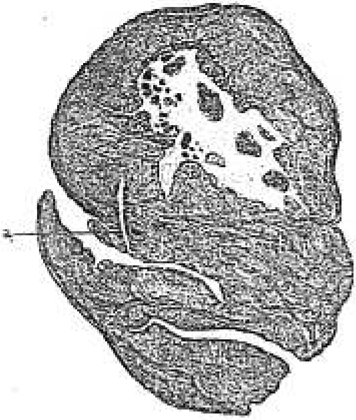
[1] Zeitsch. f. klin. Med., 1896, No. 30, p. 529.

[2] Centralbl. f. Laryngologie, 1895, No. 11, p. 901; Baumgarten's Jahresbericht, No. 10, p. 454.

This case was a boy, aged twelve years, who came into the clinic of the Manhattan Eye and Ear Hospital, in the service of Dr. Lewis A. Coffin, for enlarged tonsils. They were cut out by Dr. W. N. Hubbard, one of the assistant surgeons, and the boy went on his way rejoicing, was well a month later, and still remains so, no metastasis having as yet shown itself.[1]

FIG. 1.



Camera lucida drawing of a section of a tonsil, showing under low power an abscess cavity containing masses of actinomyces. At *a* is seen the beginnimg of the growth of a colony in one of the lymph nodes. There is a larger number of lymph nodes than usual in tonsillar hypertrophy.

In the routine of the clinic these tonsils were put in formalin and later in alcohol, embedded in celloidin, cut in sections, and stained in hæmatoxylin-eosin. Subsequently the whole tonsil was sectioned, but only the one cavity was found. With the various microscopic appearances of that we are here concerned.

In Fig. 1 it will be seen the section traverses the whole tonsil covered with the usual striated squamous epithelium, with little or no keratosis. What is apparently an abscess cavity first attracts attention. It occupies a comparatively large area in the tonsil. In its cavity are seen the peculiar striated kernels which have at their centre a blurred appearance and a yellowish tinge at the surface, taking the hæmatoxylin stain very faintly. A closer examination reveals the striations more marked at the edges. (The artist has drawn them somewhat schematically in this figure.) They are seen not only free in the cavity, but less distinctly along its walls, the granular masses making the picture indistinct.

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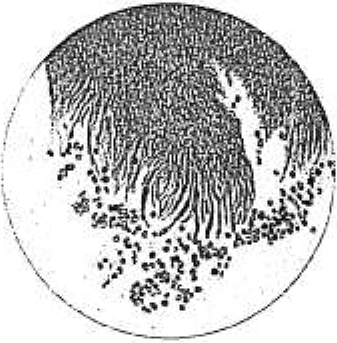
[1] I am unable to conjecture whether Buge's cases were actinomycosis or not. In view of the variations in the toxicity of the different members of the group with the same morphology and, indeed, from the clinical histories of such cases, excision of a focus, even when only moderately complete, frequently prevents recurrence.

Under a higher power there is seen in Fig. 2 the edge of one of these striated clumps and the peculiar branching, club-shaped rays of the actinomyces group appear, and through the blur and granulation of the interior of the clump one can make out the lace network, which is so characteristic. Around the ends of the ray or terminal filaments, which are variously described as loops and club-shaped

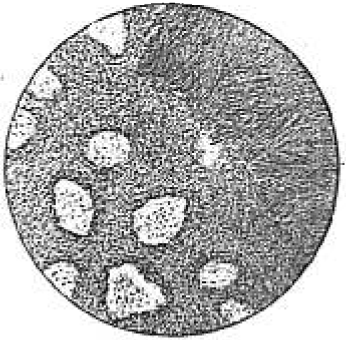
FIG. 2.



Camera lucida drawing under high power of the edge of one of the masses of actinomyces lying in the abscess cavity. The drawing does not give very accurately the "granulation" of the mass.

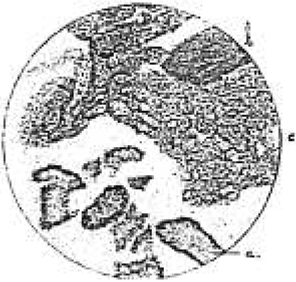
ends, are to be seen groups of much swollen leukocytes whose nuclei are granular or fragmented. When we come to examine the edge of the cavity we find that it is lined with a very greatly proliferated epithelium. In Fig. 3 it is seen that this proliferation differs markedly from that seen in crypts filled with the leptothrix. There is here no appearance of keratosis, but the fringe of epithelium is faint and very deep, enclosing islands of connective tissue. The nuclei of the epithelial cells are also fragmented. It has the appearance of the epithelium around an old syphilitic or lupoid ulcer. The ends of the actinomyces are pointed directly into the epithelium and invading it, apparently, as it breaks down by granular degeneration. Yet evidently the epithelium continues to proliferate under some stimulus, encroaching upon and surrounding islands of connective tissue, which doubtless at first contained round lymph cells. These apparently gave way first to the "granulation" process, to which the reticulum is also succumbing.

FIG. 3.



Camera lucida drawing under high power, showing the hyperplasia of the epithelium of crypt and the "granulation" due to the influence of the adjacent clump of actinomyces.

FIG. 4.

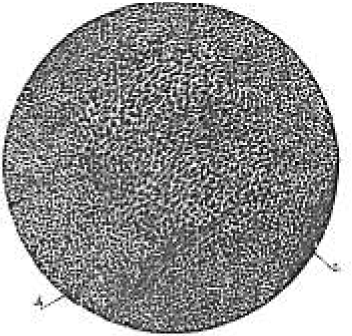


Proliferation of the epithelium of the crypt. The degeneration and the granular condition of the cells renders the epithelium atypical and indistinct. *a.* Actinomyces clumps. *e.* Epithelium. *l.* Lymphoid tissue.

It is apparent therefore that we have here the infecting germ lodged in a tonsillar crypt. After a longer or shorter time it began to multiply and excrete a toxin which, while it stimulates the proliferation of the bordering epithelium, gradually leads to its disintegration at the surface. The cavity of the crypt is thus painlessly enlarged, as there is no pressure, and the epithelium encroaches on the subjacent tissue. It is regularly the history of these actinomycotic abscesses wherever they form that they produce no pain. Neither do they form much pus, and the infection is not a mixed one. It is safe to believe that this proliferation of the epithelium has some influence in keeping the actinomyces from invading the subjacent lymphoid tissue, and we may thus understand why, without an abrasion, skin infection is such a rare occurrence.

In the warmth and moisture of a tonsillar crypt in this case this was an insufficient protection, for if you will refer again to Fig. 1 you will perceive that the lymph nodes are more swollen and prominent in the section than usual, and that in one of them at a distance from the affected crypt there is seen at *a* an area small in size. Under the microscope it has the same blurring and the same faint yellowish tinge with the low power as do the clumps in the crypt.

FIG. 5.



Camera lucida drawing under high power of the colony seen at *a* in the low-power drawing of Fig. 1. The fragmentation and breaking down of the lymph cells around the bundle of mycelial threads is very evident. *b*. Branching of the filaments. *c*. Edge of the lymph node.

When we examine this spot with a high power (Fig. 5) we see some parallel threads, without joints, but with a tendency to branch. You will note that at *b* there is no fragmentation at the ends of the small branch, but at the terminal points of the larger and chief bundle you will note a destructive fragmentation of the nuclear material of the lymph cells. Elsewhere in the node these are normal.

A great deal has been written both as to the disease actinomycosis in man and in cattle, and as to the morphology and classification of the organism with which the disease is associated.[1] While it is said by Israel that von Langenbeck observed the disease in 1845, and various others from time to time referred to the condition, its nature was not recognized until the rise of modern bacteriology. Since then various names have been suggested for the organism. It has been called a streptothrix, cladothrix, oöspora, but the name actinomyces, suggested by the botanist Harz, has remained on the whole the one usually employed. MacCallum and others have used the affix asteroides to designate the appearance of the clumps, or asterisk-like "drusen." It was Bollinger[2] who first observed, in 1876, a microphyte in the lumps which are so often seen about the jaws of cattle, and referred the organism to Harz for a name. In the same year Israel described the disease in man,[3] but did not connect it with the cattle affection. This was pointed out by Ponfick[4] in 1879. Classified at first among the fungi, it was supposed to be insusceptible of artificial culture. Although Johne failed in 1882,[5] a more successful attempt was made by Boström in 1885.[6]

Since then numerous and varied experiences with its life forms and different culture media have led to much discussion.[7]

I do not propose—indeed I am not able—to refer to all the variations in the pleomorphism of the organism. Suffice it to say that it appears under various conditions as a streptothrix or streptobacillus, long threads sometimes straight, sometimes in loops, sometimes in such short rods that they resemble cocci. Club-shaped branching appearances are shown in my drawings. As I understand it, the question of spore formation has not been fully settled. Mertens[8] declares that their presence has not been proven, while the contrary is asserted by Berard and Nicolas (*ibid.*).

It seems the drift of recent opinion that "a number of different micro-organisms may cause the typical disease picture."[9] Not only is this the opinion of Dr. W. T. Howard, but more recently[10] we are to infer from the researches of the same observer, supported by the testimony of others, that there are varieties, indistinguishable morphologically from pathogenic forms, which are entirely harmless and non-pathogenic in animals, Howard having found such fungi growing in various samples of vaccine.[1]

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[1] A reference to the indices of the Centralblatt f. Bakteriologie, the Centralblatt f. Laryngologie, and Baumgarten's Jahresbericht will put anyone on the track of practically all that has been written on the subject in twenty-five years.

[2] Centralblatt f. d. med. Wissensch., 1877, No. 27.

[3] Virchow's Archiv, Bd. lxxiv.

[4] Berl. klin. Woch., 1879, p. 347.

[5] Deutsche Zeitsch. f. Thiermed., 1882, Bd. vii.

[6] Berl. klin. Woch., No. 1.

[7] The latest contribution I have seen being the article on the subject by Lignieres and Spitz, under the title "Contribution à l'étude à la classification et à la nomenclature des affections connues sous le nom d'actinomycose." Centralbl. f. Bakt., I. Abt. Originale, 1904, No. 3, Bd. xxxv.

[8] Centralbl. f. Bakt., 1901, No. 16, Bd. xxix.

[9] Howard. Actinomycosis of the Central Nervous System, Journal of Medical Research May, 1903.

[10] Ibid., January, 1904.

As yet it remains undecided whether these varieties can be distinguished by culture methods, and at present, so far as I know, observation of inoculation of animals and a study by culture methods of cases occurring in man furnish the most promising fields of differentiation. It is only fair to say that so recent a writer as Mertens[2] agrees with the earlier statements of Pfeiffer that the division of actinomyces even into two varieties seems unwarranted, but it would seem from the work of Silberschmidt (quoted by Howard) and Lignières and Spitz that this opinion is now untenable, Howard quoting Lachner-Sandoval to the effect that there are as many as twenty-five varieties of ray fungi.

As to the tinctorial reactions: "Mycelium and spores stain by Gram and retain carbol-fuchsin after treating with 20 per cent. sulphuric acid." "In sections stained with hæmatoxylin or methylene blue the colonies are finely granular, and stain homogeneously with the blue stain" (Howard).[3] On the contrary, MacCallum[4] says: "The staining is not regular or homogeneous, and one frequently finds clear unstained areas, almost like spores in some bacilli, or, on the other hand and more commonly, only granular bodies along the course of the filaments take the stain, so that the organism looks like a dotted line, sometimes with a faint outline on each side of the row of dots. These fine grains are irregular in form and do not suggest spores."

Other observers also differ in so far as they look upon some of the granules as spores or cocci forms or fragments of degeneration.

A very much more interesting and directly suggestive part of the subject is the conclusion toward which a study of its morphology and its culture has led many observers as to its classification and its affinities to other organisms. Some of its pathogenic characteristics, as we shall see later, are also, to say the least, very suggestive in this respect.

To quote again from MacCallum: "The organisms show affinities on the one hand with the bacteria and on the other with the fungi. . . . Their size approaches more nearly that of the bacteria than that of most fungi, and they stand nearer to the bacteria than to the fungi from the fact that their filaments do not, as in the fungi, consist of tubules with doubly contoured wall and divided into segments by septa. They resemble the fungi, however, in being branched structures which produce under certain conditions fine conidia on their terminal filaments."

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[1] It is quite possible that Ruge's cases were really the actinomyces which had failed to penetrate the tissues, owing to their feeble toxicity toward their hosts. In my own case, though growing in a lymph node at one place, the organism may have been but feebly pathogenic.

[2] Loc. cit.

[3] Loc. cit.

[4] Centralbl. f. Bakt. Originale, 1902, No. 12, Bd. xxxi.

We may omit here any extended reference to its affinities morphologically with the Klebs-Loeffler diphtheria bacillus. The subject has received some attention from Silberschmidt[1] and others.

A much more interesting subject for consideration is its relationship to the tubercle bacillus. As long ago as 1888 Metschnikoff drew attention to the variations in the morphology of the tubercle bacillus, and Czaplewski still further elaborated the subject in 1891. Metschnikoff declared that the tubercle bacillus was not the final stage, but represented a condition in the developing cycle of a thread-like bacterium. He showed the branching of the bacillus and the bud-like projections sometimes seen even in human tuberculous sputum. Mafucci demonstrated the same appearances more often seen in fowl tuberculosis. Fischl, in 1893, showed that these forms occasionally occurred in pure cultures, and seems to have been the first to call attention to the resemblance of the bacillus in this form to the actinomyces. A. Coppen Jones,[2] from whose work I have quoted the above, in 1895 pointed out characteristics common to both the organisms, and the details are beautifully shown in his plates. Pettersson[3] in 1899 drew attention to the culture affinities of the acid-resisting bacilli, and more recently Abbott and Gildersleeve[4] have again returned to the subject and remarked upon the "actinomyces-like development of some of the acid-resisting bacilli." It must be remembered that, besides various modifications in the pathogeny of the tubercle bacillus itself, the human, the bovine, the fowl, the fish, the turtle tubercle bacillus, there are the morphologically and in cultures closely allied grass, hay, and butter bacillus, which have no pathogenic properties at all, though the above authors have shown a slight amount of tissue change occurring when they are injected. Indeed, so far as pathogenesis to man is concerned, we have in the group a steadily progressive series of organisms from the harmless butter bacillus of Rabinowitsch, through the turtle and fish bacilli,[5] to the bovine and human bacilli, concerning the mutual virulence of which war has lately been waging

We know with much less accuracy the interrelationship, as to pathogenesis, of the various members of the actinomyces group of bacilli, but we have some reason to believe that a similar series will be revealed in this family of organisms. Abbott and Gildersleeve have shown that the grass and hay bacilli injected in animals form in clumps with granular leukocytes around them in a manner similar though not identical with the actinomyces clumps.

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[1] Deutsche med. Woch., 1901, No. 47.

[2] Centralbl. f. Bakt., Nos. 1 and 2, Bd. xvii.

[3] Berlin. klin. Woch., 1899, No. 26.

[4] Centralbl. f. Bakt. Originale, 1902, 1st Abt., Bd. xxxi.

[5] Friedmann, Centralbl. f. Bakt. Originale, 1903, Nos. 7 and 8, Bd. xxxiv.

Finally, the actinomyces is essentially an aerobic organism, and it has been shown by Coppen Jones, most recently supported by Wollbach and Ernst,[1] that the tubercle bacillus growing on a favorable medium shows more frequently the branched forms when the access of oxygen to the culture is most free. Whether the ray-fungus, on the contrary, shows more forms analogous to the usual forms of the tubercle bacillus when its culture medium is poor and the access of oxygen limited, I am unable to ascertain. Suffice it to say, as to classification, that generically these two microphytes seem closely related and stand between the lower and higher forms of microbic life. To use expressions current in German literature, the actinomyces is a "facultativ-parasite," the tubercle bacillus a "facultativ-saprophyte," *i. e.*, they both grow, though with difficulty, the former within and the latter without the animal body. Conversely the actinomyces grows readily on plants and the tubercle bacillus readily in animals.

It is necessary to say a word of the occurrence of actinomyces in nature and of the source whence man derives the organism when infected. It was formerly surmised, after Ponfick had shown the disease in man was identical with the more common infection in cattle, that man derived it direct from the latter, and, as usual, it was claimed in Germany that American cattle were more commonly affected than the beasts of the Rhineland. As a matter of fact, however, the majority of cases in which it was possible to trace the infection from an external source have shown that the organism was carried into the tissues, usually of the mouth, but rarely of the skin,[2] by the intrusion of foreign bodies, such as stalks and ears of grain or pieces of wood. Indeed, grain kernels have been found between the teeth of cattle, on which actinomyces were growing. It has been shown by Berestnew that straw dampened and placed in a thermostat will frequently, after a week or two and the removal of other more quickly growing colonies, show the presence of the actinomyces. The organism grows in the comparatively large air cavities and channels of the straw stems, doubtless because it is left there unmolested, yet exposed to the air a sufficiently long time to fructify and draw sustenance from the plant cells. It would seem therefore a matter of considerable doubt if the germ enters the system of man very frequently with the meat of cattle, though the number of cases of primary intestinal or peritoneal lesions might argue in favor of this method of infection. At the most it is a rare affection in man in any situation.

In discussing the port of entry of the organism we find another field in which something may be gleaned of interest in the wider topic of human tuberculosis. It is perfectly apparent, in looking through the abstracts of cases reported in laryngological literature, that reports in the vast majority of the cases refer to lesions about the jaws and necks, many of them proved to be connected directly with a carious tooth cavity. Naturally the criticism is suggested that laryngological literature is hardly the place to seek this sort of evidence of the comparative frequency of the location of the lesion. We will therefore turn to bacteriological literature.

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[1] Journal of Medical Research, December, 1903.

[2] Von Bergmann, Berl. klin. Woch., 1904, No. 1.

In 1887 Bollinger[1] stated that Moosbrugger, in collecting reports of ninety cases, found that in 10 per cent. no port of invasion could be made out, but in the majority of cases the mouth and pharynx seemed to be the starting point.

On glancing at the cases referred to in the *Centralblatt für Bakteriologie* since 1887 a tally, which may not be absolutely correct, shows that the primary lesion was situated around, or the port of entry of the infection could be traced to:

The neck or jaws, mouth or pharynx	72 cases.
Primary lesion in abdomen or its viscera	18 "
" " thorax walls, lungs, or pleuræ	21 "
" " brain	3 "
" " skin	3 "
Total	117 "

In 1899 Rütmeyer,[2] from a review of the literature surmised that the port of entry was the mouth and pharynx in 50 per cent. of the cases; lungs, 20 per cent.; intestines, 15 per cent. Of the 113 cases studied by Miller[3] in the same year, 53 were referred to the regions of the mouth and pharynx.

In a very large number of cases the tissue changes can be traced directly to a carious tooth cavity. In others they stand in such relation to a carious tooth that the port of entry can hardly be doubted. It is true many infections occur about the jaws of people with healthy teeth, but in those of untidy habits the crevices between the teeth, as we have seen in cattle, would furnish a sheltered nook for the slowly noxious effect on the epithelium. A careful analysis of the cases will show that the larger number of cases of lesions about the jaws present a lesion about the *lower* jaw, the lesions about the upper jaw being in a marked minority. We can scarcely explain this, when we have such strong evidence that the teeth are at fault, except on the assumption that the force of gravity drains the tooth cavities and normal crevices between the teeth of the upper jaw. While of course an imperfect protection, it would be sufficient to account for the greater preponderance of apparent infection through the teeth of the lower jaw. I desire to lay emphasis on this influence of gravity on the portals of infection, as I shall return to the subject when we come to consider the bearing of the evidence of this rarer form of bacterial infection upon the more important subject of tuberculous infection.

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[1] Centralblatt f. Bakteriologie, 1888, p. 501.
[2] Berl. klin. Woch., 1889, Nos. 3 and 4.
[3] Die Mikro-organismen der Mundhöhle, Ed. 1889.

Evidence of the influence of gravity is still further presented in the rarity of infection with actinomyces through the skin. This has occurred, but so far as I know the proof of its actual occurrence has, with one exception, always been associated with the entrance of a foreign body. In 1895 Hummel,[1] in a search of all literature, found but twelve cases, to which he added one, in which it seemed that the organism entered the tissues in a breach made by a foreign body, the grain of a cereal, in all but two cases. The case related by von Bergmann, in which actinomyces infection followed the kick of a horse, may be placed in the same category, but here the solution of continuity was in the skin, while in the other cases referred to the mucous membranes were pierced in the majority of the reports. While therefore we have good reason to believe that man, like cattle, receives his actinomyces infection in his food on grain and vegetables rather than in his animal food, it seems probable that the organism must either be carried at once beneath the epithelium by the intrusion of a foreign body, or it must find some lurking-place where it may remain in contact with the epithelium for a long space of time under the influence of warmth and dampness.

The query arises, Could it under such circumstances pass through the intact squamous epithelium? If you will glance at Figs. 3 and 4, you will see what effect it is exerting on such epithelium, while the evidence of Fig. 5 will convince you that somewhere in this region of the tonsil it has passed the epithelial barriers. In support of this evidence of a microbe allied to the tubercle bacillus being able to pierce squamous epithelium even of the skin, when placed under proper conditions of warmth and dampness, I may cite the instances referred to by von Bergmann where actinomyces caused lesions in the crevices of old scars, as in those from breast operations, too long after operation, presumably aseptic, to think of entrance at the time of the skin cutting. This might be a profitable point to clear up by animal experimentation, making artificial skin pockets and placing in them actinomyces of known virulence.

It is difficult to say how the chemistry of the gastrointestinal tract affects the actinomyces. It is of course certain, considering its prevalence on plants, that it is frequently carried into the stomach of man with his food, and still more frequently into the gastrointestinal tract of cattle.[2] I am not acquainted with any investigations which show the relative frequency of actinomyces disease of the abdominal organs of cattle. I suspect, from general laws, that such lesions are relatively rarer in cattle than in man. As we have seen, abdominal actinomycosis stands next in frequency to that of the lungs in man. It is very probable that many cases of apparent primary pulmonary actinomycosis are secondary to lesions higher up which have not been recognized, but whatever may be the truth in regard to tuberculosis, I think we can hardly thus explain the cases of actinomycosis of the abdominal cavity. In reference to this point Grill,[1] who in 1895 collected 107 reports of cases of intestinal actinomycosis, considered the germs too big to be carried by lymph channels, a statement which will hardly stand criticism to-day, though it would seem that *something* hinders them from travelling much by the lymph channels. It seems likely they are carried chiefly by bloodvessels.[2] Although no part of the intestinal tract seems to be proof against their absorption, most of the cases proceed from the cæcum and the vermiform appendix. I have noted three reports of appendicitis due to it. This would be a locality where a germ would lie longest undisturbed. I wish to draw attention to the rarity with which not only actinomycosis but tuberculosis seems to have its origin from œsophageal absorption, which can scarcely be accounted for in any other way than by the rapidity with which the infecting material passes the surface. The same may be said in regard to nasal infection. The constant drip of secretions from the upper regions of the nose tends to wash rapidly away any germs deposited in the lower respiratory channels of the nose. The sluggishness of the lower bowel gives an opportunity for the absorption of bacterial ptomaines and of the bacteria themselves, which is not presented by the upper digestive tract, due doubtless in large measure to its active peristalsis. Within the tissues we have an instance of the opportunity which stasis offers the tubercle bacillus in the preponderance of primary apical lesions of the lungs over that of other pulmonary localities.

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[1] Centralbl. f. Bakt., 1895, No. 2, p. 180.

[2] In this, however, I may be mistaken, for Mari (Centralbl. f. Bakt., 1892, Bd. ii. p. 854) examined for actinomycosis 42,230 cattle and found the lesion in 1030, or 0.02½ per cent. of the total number. Of these 1030 cases the lesion was about the lips in 621, or about 60 per cent., which as we have seen is not far from the ratio about the mouth in man. Berg (Centralbl. f. Bakt., 1899, 2te Abt., Bd. xxvi. p. 231) is said to have examined 400,000 (?) sheep and found the lesion only in three cases, and these about the mouth.

From the consideration of these *a priori* reasons, supported to some extent by more direct evidence, the inference has been that the faucial tonsil, standing at the very forks where the breathway and foodway meet, existing normally in the form of folds and pits, but these pockets exaggerated enormously in hypertrophic disease, is the spot par excellence whence all sorts of pathogenic bacteria would start on their maleficent journey through the blood and lymph channels of the human body. From this alone we might expect to find there protective influences which prevent the annihilation of the race. More than forty years ago Virchow declared it to be entirely incomprehensible why the tonsils possess immunity in spite of the fact that they are lymphatic organs. We have learned much since then, but in spite of the horde of names recently poured into the already gorged channels of pathology by the recent researches in immunity we all recognize that we know comparatively little of protective influences. We only know they are multitudinous, and gathering them together into one word, "immunity," is convenient, but it has been very misleading. I think Figs. 3 and 4 show that the squamous epithelium lining the crypts of the tonsils is one of these protective agents against the invasion of the actinomyces. Now I have hinted that forces which keep the bacteria on the move, dripping secretions, waving cilia, intestinal peristalsis, gravity, must also be placed in the category of the defenders of the animal organism against the invasion of the pathogenic germs, these being more important in the production of that relative local immunity to which Virchow so often referred. That this keeping the germs on the move plays no inconsiderable part in slowly growing germs like the actinomyces and the tubercle bacillus, preventing them from penetrating the epithelium, we may readily believe if we are warranted, as seems the case with the actinomyces, in thinking that their toxins play a part in opening a port to their intrusion.

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[1] Centralbl. f. Bakt., 1895, 2, p. 181.

[2] This suggestion is supported by the description of the post-mortem findings in a case reported by Lesin (Centralbl. f. Laryng., 1835, No. 11, p. 901). It is probable, as I have said, that in this case the port of entry was the tonsil, since the primary lesion was in this vicinity. There was much infiltration around a large bloodvessel in the neck, and the case terminated fatally from cerebral involvement.

But if a tooth cavity will lodge a slow-growing germ long enough why will not a tonsillar crypt? And if so, why does not actinomycosis of the tonsil more frequently occur?[1]

One reason is doubtless, as we have seen, the resistance of the epithelium. Such protection does not obtain in a carious tooth cavity. There are other protective influences which are associated with the tonsil, and not with dental caries; these are gravity, mobility, exosmosis.

In speaking above of the drip of secretions rendering the upper meatuses of the nasal cavity relatively aseptic and the inferior meatus less foul than we would expect, I have intended to introduce the consideration of a factor which must obtain on all moist surfaces of the airway and foodway to some extent. The excretion of fluids, osmosis from within outward, whether through glandular or surface epithelium, must be a process which tends to wash out or keep out the pervasive germ. In the absence of the waving cilia of the epithelium, which works against gravity only in the dependent cavities, such as the accessory nasal sinuses and the pulmonary tract, gravity carries the secretions and their bacterial contents downward. These secretions themselves are apparently to some extent inimical to the existence or rather to the continued virulence of pathogenic germs, but the gastric secretions annihilate many of the bacteria carried into the stomach. In addition to exosmosis, which, since it has not been demonstrated for the tonsillar crypts, we may for the moment ignore, the motion imparted to the tonsillar structure by muscular action in deglutition, as in intestinal peristalsis, is sufficiently evident. The unskilled examiner is fully aware how, when he nervously forces down the tongue, the large tonsillar crypts gape in the reflex gagging of the patient. In the act of deglutition the passage of fluids and masticated solids across the mouths of gaping tonsillar follicles must tend to suck and siphon out the contents of the crypts and lacunæ. It is true of course that the configuration of some of these is such that they are thus incompletely emptied, but the conception that these cavities are the constant and crowded lurking-places of all sorts of bacterial life has been very much exaggerated. I have stained many hundred sections of tonsils to demonstrate in them the presence of bacteria. Not only have I failed in all but an insignificant number of cases to detect any bacterial forms beneath the deeper layers of the epithelium, but I have been much more puzzled to account for the comparatively few crypts which show any considerable number of bacteria outside of the epithelial wall. In very sparse numbers, it is true, with diligent search, they may be found if properly stained. Here and there one or possibly two and three cocci may be seen lodged in a nook under the upturned edge of an epithelial cell. This is the general rule, but it occasionally happens that vast numbers may be seen in a crypt, in striking contrast to neighboring lacunæ. I can only conclude that the complete or partial closure or the valve-like action of the mouth of the crypt has prevented the cleansing of the cavity in the manner I have described. These are the crypts recognized clinically by the white, cheesy masses in them, but they are insignificant in number when compared to the total number of recesses. The mechanism which keeps the latter free of visible solid matter is also a very efficient scavenger of bacteria.

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[1] The suggestion of Westenhoeffer (Berliner klin. Wochenschrift, 1904, No. 7) that the most frequent port of entry of the tubercle bacillus in infancy is through the break in the epithelium of the gums at the time of dentition, does not suffice in accounting for the usual route of entry, yet both in this breach and in the cavities of carious teeth the bacillus tuberculosis may find that opportunity which seems to furnish the most frequent access to the actinomyces.

Nevertheless there are enough germs in contact with the epithelium for a sufficient length of time to force ingress were they endowed with powers of penetration, but usually these bacteria are either non-pathogenic, or if they are pyogenic cocci they are non-virulent toward their host, as long at least as the latter's systemic repelling forces are in proper order. In such a swarm of microphytes as I have just alluded to a slow-growing tubercle bacillus or an actinomyces would ordinarily have an unsuccessful struggle for existence. We are fully warranted in believing that thus non-pathogenic parasitic bacteria exert at the so-called portals of infection a protective influence over the animal organism, a presumption which under other phases is a familiar and well-accepted belief.

But why is it that under such conditions of abundant bacterial life in a crypt—the cavity fairly gorged with microphytes—we can detect none beneath the epithelium? We are familiar with the experiments of Goodale and Hendelsohn which have shown that minute carmine particles injected into the crypts or even simply blown on the surface readily pass through the epithelial barriers. Pirera[1] performed similar but not very convincing experiments with the bacillus prodigiosus, but his results are so diametrically opposite to what we observe in actual occurrence of plugged crypts that I am not disposed to accept his conclusions that bacteria also pass through the epithelium.

It is true that in acute inflammation staphylococci, streptococci, diphtheria bacilli are to be seen in the subepithelial structure. It is exceedingly doubtful if at such times we can suppose that other slow-growing forms (*e. g.*, tubercle bacilli) survive in the struggle for existence in the crypts and live to enter the tissues. In the quiescent tonsil the picture is quite a different one, and if we are to suppose that it is then the tubercle bacillus enters, it evidently differs from the other bacterial contents of the crypt.

Distrusting experiments in which a mass of powdered substance in exceptional fashion was deposited in the crypts and blown on the surface of tonsils, I studied numerous unstained sections. I sent to the dusty city of Pittsburg, and through the great kindness of Dr. Day and Dr. Jackson I was able to secure the tonsils of some of its citizens, exposed for years to the deposit of coal-dust particles. There can be no doubt that dust thus deposited in the usual way follows the same course as that deposited in the experiments of Goodale and Hendelsohn. It seems therefore that the dust passes, but cocci and bacilli of the same or smaller dimensions do not pass into the quiescent tonsil. At least if they pass they do so in such greatly reduced numbers that it leaves the contrast to the behavior of dust particles a striking and unexplained phenomenon. There are certain cases which are very rare, but which we nevertheless occasionally see in the clinics, in which are to be observed fine white points scattered throughout the tonsil itself, which are not plugged crypts. Microscopic examination will frequently show these to be tiny abscesses of the lymphoid tissue, small cavities gorged with cocci. Further examination shows that numerous cocci may be seen elsewhere in the lymph channels of the tonsil at a considerable distance from these foci.

Without pain or fever or constitutional disturbance of any kind it is evident that something has occurred here which has permitted the entrance of the surface bacteria. The cases I have seen have been in children with a phthisical family history, and the thought suggested itself to me that these individuals may also absorb dust more readily than the numerous other cases I have studied in which I made no selection. I was fortunate after some time to receive from Pittsburg tonsils cut from children with a known marked tuberculous family history and from cases of phthisis pulmonalis. Neither in these nor in the lymphoid abscess cases could I make out any greater amount of dust passing.

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[1] Archivii Italiani di Laryngologia, Fasc. 2, 1900.

We are free to infer that nature in the process of repelling the invasion of morbific germs has in the process of evolution accumulated around these diverticula of the tonsils, as elsewhere in the alimentary tube, those cells of the organism which supply the antibodies to the tissue juices, and that they are held in place by the mesh of lymph channels which may be too small at places to transmit some germs (*e. g.*, the actinomyces). This at present is a familiar conception, but from the study of the problem as indicated above we seem to be also in the presence of some wonderful mechanism of nature, evolved through æons of time, and established at the portals of infection, by which harmless atoms glide through the stomata of cells, or their interstices, which close at the approach of the microbic enemy. When that mechanism is in perfect working order we may conjecture that the toxin of the actinomyces, the tuberculin of its more deadly cousin, is powerless to overcome it. These germs may enter immediately the undefended portals of the "predisposed," or they may lurk at the door until, in an unguarded moment—at the moment of exhaustion from illness, starvation, or other systemic disturbance—the toxin opens the path and the microphyte passes the first barrier. I do not know what part, if any, the epithelium of the tonsils may play in the production of antibacterial ferments. It is supposed by some observers that the intestinal epithelium is an important tissue in its manufacture. If so, the problem in the crypts of the tonsils may also be a chemical one, but it seems to me the attention of observers has been too intently fixed upon what follows after the microbe has passed the epithelial barrier. It is the error we are constantly falling into, the exaggeration of a single factor in the body's fight against disease. We forget that the etiology of disease is a complex problem.

I do not know how much resistance is offered by the normal epithelium to the tubercle bacillus. I have definitely shown that under morbid conditions it may pass freely through squamous epithelium.[1] You will see by a glance at Figs. 3 and 4 how the tonsillar epithelium reacts to the presence of the adjacent actinomyces. I am sure you will be struck by the resemblance to the epithelium around ulcers, especially of a syphilitic or tuberculous nature. Now after the actinomyces has passed at some unguarded gate you see how its toxin has fragmented the nuclei of the lymph cells in Fig. 5, and again we are reminded of the specific granulomata.

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[1] See the plate attached to the paper on "Tuberculous Infection of the Lymphoid Tissue in the Pharynx, with some remarks on Laryngeal Infection," New York Medical Journal, September 26, 1896, read that year before the American Laryngological Association.

There is another curious analogy to syphilis at least. Van Niessen's[1] observation of the occurrence of a microphyte resembling the actinomyces in cases of syphilis has, so far as I know, received no support, but not only has the iodide of sodium a very favorable effect upon the lesions of actinomycosis, but solutions of comparatively weak strength (⅛ per cent.) retard and stronger (½ per cent.) solutions inhibit the growth of the actinomyces.

Whatever may subsequently be proven to be the virus of syphilis, we know the effect on cells under certain conditions of the virus of actinomyces and of the tubercle bacillus. What we have seen and what we are familiar with is the result of a destructive agent upon cell life. We know very much less accurately the biological processes which the toxin of these bacteria excite in the activity of the cells. The tuberculin evidently profoundly modifies their function. What it does in a chemical or biological way is shadowed forth in some of the terms in the recent observations on immunity, but how it affects them mechanically is entirely unknown. How the endothelium of the lymph channels may dilate or contract or become swollen we do not know, and yet that lies at the root of the problem of their carriage through the tissues. In this connection a recent remark of von Behring is suggestive:[2] "To scrofula I ascribe the alteration in the muscular coats of the blood-vessels brought about by T. B. infection, which finds an expression in excessive sensitiveness toward tuberculin, and which in general may be compared to the scrofulous diathesis of the older writers."

To pursue this subject farther would lead us too far from the portals of infection, which was the site of the activities in this unique case of actinomycosis of the tonsil.

REPORT OF A CASE OF RUPTURE OF AN ANEURYSM OF THE AORTA INTO THE LUNG, WITH SPONTANEOUS RECOVERY.

By N. S. Ferry, M.D.,
OF MEMPHIS, TENN.

The following rare case occurred in the service of Dr. A. R. Defendorf, pathologist and assistant physician to the Connecticut Hospital for the Insane, with whom the writer had the pleasure of working during the summers of 1900 and 1901. The history of the case was kindly furnished by Dr. Defendorf.

The patient was a man of unknown personal history, except that he had had syphilis, suffered from organic dementia, the onset of which occurred at forty years of age, immediately following head

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[1] Berl. klin. Woch., 1904, No. 4.

[2] Centralbl. f. Bakt., 1898, No. 2, Bd. xxiii.